Evaluation of Dermal and Respiratory Chloroform Exposure in Humans

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Chloroform is a known contaminant of chlorinated drinking water and of swimming pool water disinfected with chlorine or one of its derivatives. Few data exist regarding the importance of dermal and inhalation exposure routes to the chloroform body burden resulting from domestic and recreational use of chlorinated water. In our experimental study involving 11 male swimmers, we quantified the body burden resulting from exposure to various concentrations of chloroform in water and air of an indoor swimming pool, during a daily 55-min exercise period. From the first to the sixth exercise period, CHCl₃ mean concentration in water was increased from 159 µg/l to 553 µg/l. Corresponding mean air CHCl3 level ranged from 597 ppb to 1630 ppb. To dissociate the dermal exposure route from that of inhalation, swimmers used scuba tanks during an additional exercise period. Chloroform concentrations were measured in alveolar air before and after each exercise period, as well as after 35 min of physical activity. Chloroform levels in water and air were measured every 10 min. We examined the relationship between alveolar air concentration (a measure of body burden) at 35 and 55 min and environmental chloroform concentrations by using multiple regression models. The natural logarithm of alveolar air concentration was strongly correlated with aqueous chloroform concentration both at 35 (p < 0.001, $r^2 = 0.75$) and 55 min (p < 0.001, $r^2 = 0.86$). The relationship with air concentrations was also statistically significant (35 min: p < 0.001, $r^2 = 0.58$, 55 min: p < 0.001, $r^2 = 0.63$). Two other variables, intensity of exercise and physiological characteristics of the subject, were also significantly associated with variation of body burden. Approximately 24% of body burden resulted from dermal absorption. Swimming in indoor pools may be an important source of exposure to chloroform. Key words: alveolar air, chloroform exposure, swimming pools, trihalomethanes. Environ Health Perspect 102:1082-1087 (1994)

Trihalomethanes (THMs) are volatile halogenated hydrocarbons which are formed during water chlorination (1–4) through reactions involving natural organic substances present in raw water. Major THMs include chloroform (CHCl₃), bromodichloromethane (CHCl₂Br), dibromochloromethane (CHClBr₂), and bromoform (CHBr₃) (5). CHCl₃ is generally the

most abundant of all THMs, and its toxic effects have been studied extensively. Because CHCl₃ has been shown to be carcinogenic in laboratory animals (6,7), there is concern over long-term human exposure to this solvent.

The total amount of THMs in a swimming pool is related to the concentration of THMs already present in water and the quantity of THMs produced in the swimming pool itself (8). For the latter, the principal variables are chlorine dosage, concentrations of organic precursors (principally of human origin) and bromide ion concentration, resulting essentially in the formation of CHBr₃ (8). In freshwater swimming pools, CHCl₃ is clearly the most abundant contaminant.

Several authors have documented significant quantities of CHCl₃ in swimming pools (8–14). In general, concentrations varied between 50 and 300 μ g/l. However, some pools contained levels ranging between 700 and 1000 μ g/l (8).

Because CHCl₃ is volatile, it will also be present in ambient air of indoor swimming pools. Lahl et al. (8) documented concentrations varying between 36 and 241 μg/m³ in the air of eight indoor swimming pools in Germany, whereas Aggazzotti et al. (12,14) noted levels ranging from 410 nmol/m² (51 µg/m²) to 5445 nmol/m³ (680 µg/m³). In addition to the concentration of CHCl₃ in water, CHCl₃ levels in indoor swimming pool air will be influenced by the number of swimmers and the turbulence caused by their movements, water and air temperature, as well as the amount of ventilation (8). Air concentrations are greatest at the water surface and decrease progressively as the distance from the water increases (8). Water ingestion occurs during swimming (50 ml for an adult) (8), but swimmers are mainly exposed to CHCl₃ through pulmonary and dermal routes.

Swimming is a popular activity. In Canada, it is ranked third among preferred physical activities after walking and gardening (15), and is enjoyed by 46% of the population aged 10 years and older (15). In addition, there are currently 14,000 swimmers registered with "Swimming-Canada," many of them spending several hours per week in a pool. The same may

be said for instructors, lifeguards, and maintenance personnel of indoor swimming pools. Few data exist on CHCl₃ exposure, not only for the population in general, but also for these high-exposure groups.

To our knowledge, few studies have evaluated the CHCl3 body burden from swimming. Aggazzotti et al. measured CHCl₃ plasma levels in 127 adults after they visited indoor swimming pools (12). The subjects were classified into three groups: competitive swimmers, swimmers taking lessons, and visitors. Although water levels were relatively low (18-50 µg/l), CHCl3 was detected in samples from all three groups. Values varied from 1.8 nmol/l (0.225 µg/l) to 21.6 nmol/l (2.7 μ g/l), compared to less than 0.8 nmol/l (0.1 µg/l) for a control group of 40 unexposed subjects. Plasma concentrations were related to the aqueous and environmental air levels, the number of swimmers, the time spent in the pool, and the intensity of physical activity. Aggazzotti et al. concluded that exposure to CHCl3 in swimming pools must be accounted for in evaluating total CHCl₃ exposure

Copaken (13) documented blood CHCl₃ levels in five volunteers before and after a 2-hr swimming session in a slightly contaminated pool (37.9 µg/l). On average, CHCl₃ blood levels increased by 0.61 µg/l. This author concluded that CHCl₃ may represent a risk for lifeguards and competitive swimmers who spend long hours in swimming pools (13).

None of the above-mentioned studies examined the relationship between CHCl₃ body burden and varying concentrations of this solvent in air and water. Furthermore, previous studies did not attempt to dissociate the dermal from the pulmonary exposure route in a swimming-pool setting. There are few data on dermal CHCl₃ absorption in humans (16). However, based on results of a recent study showing substantial dermal CHCl₃ absorption while showering (17), the skin may be the major entry route while swimming.

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We studied the change in CHCl₃ body burden in male swimmers resulting from various environmental concentrations in an indoor swimming pool setting. We also examined the relative contribution of dermal and respiratory routes with respect to the total CHCl₃ body burden.

Methods

Data were collected from 17 April to 23 April 1993 at an indoor swimming pool $(25 \text{ m} \times 10 \text{ m} \times 2.5 \text{ m})$ used to train scuba divers. The atmosphere of the pool was under negative pressure, and ventilation rate was kept constant during the study time. Eleven male subjects (age 19-38 years, median 23 years), members of a scuba divers association, were recruited following an information session about the project. After signing a consent form approved by the Ethics Committee of Laval University (Québec, Canada), the subjects went through a daily 55-min exercise session on seven different days. Each session consisted of three 15-min periods of swimming, separated by two 5-min intervals of rest. The intensity of physical activity was sought equivalent to 45% of maximum capacity for six sessions and 65% for one session. To isolate the route of dermal exposure from that of respiratory exposure, the subjects used scuba tanks during one session. Beforehand, air from the compressor used to fill the tanks was tested and found free of CHCl3 and the subjects did not breath air from the pool during this session.

During a pretest, we verified the homogeneity of water concentrations and validated our calculations as to the volume of pure chloroform needed to achieve the required concentrations. Then CHCl₃ concentrations in pool water were controlled. Each day, once the swimmers had left after the exercise period, the amount of CHCl₃ was increased so that a slightly higher concentration was obtained for the next period. CHCl₃ (Aldrich, Canada) was injected into the pool water using a highvolume pump (Gilian, HFS-113A) connected to a tygon tube equipped with a branch pipe and a funnel. The tube was positioned in the middle of the swimming pool and secured to the bottom with a fixed weight.

During each exercise period, we collected three alveolar air samples from subjects (before exercise, after 35 min, and at the end of the 55-min session). Differences between CHCl₃ concentrations measured at 35 or 55 min and those determined at the beginning of exposure yielded estimates of body burden resulting from swimming for the two corresponding periods (Δ35 and Δ55). Measurement of alveolar air CHCl₃ was preferred over plasma

CHCl₃ because air sampling was more practical, less invasive, and allowed for adequate sensitivity during low-level exposures. Other researchers have reported this measurement to be an excellent indicator of CHCl₃ body burden (14,17).

We measured aqueous and ambient air CHCl₃ concentrations every 10 min during the study time in the middle of the swimming pool. Air and water samples were collected respectively from the respiratory zone of the subjects and at a water depth of 20 cm.

We collected environmental samples according to Aggazzotti et al. (12). Alveolar air was sampled according to the following protocol. After taking in a deep breath and holding it for 20 sec, the subject exhaled into an inhalotherapy tube attached to a rubber fitting connected to a 30-ml sampling syringe. Exhaled air exited by two holes made in front of the piston located at the end of the syringe. Before the end of exhalation, the piston was positioned to obstruct the holes and to collect a sample volume (20 ml). Using a disposable needle (25 gauge), the sample was injected into a vial under negative pressure. Samples were sent to the laboratory and analyzed within

CHCl₃ concentrations in alveolar air, environmental air, and swimming pool water were measured using headspace gaschromatography techniques similar to those developed by Aggazzotti et al. (12). A headspace HS-40 injector (Perkin Elmer) coupled to a chromatograph (Varian Vista 6000) and an electron capture detector were used. Separation was achieved using a DB-1 1 µm, 30 m ×

0.25 mm, capillary column (J&W Scientific). Helium (14 psi) served as the carrier gas.

Detection limits determined using three times the standard deviation of the blank were 0.5 µg/l for water and 10 ppb for ambient and alveolar air. CHCl₃ concentration measurements were linear for water from 0 to 800 µg/l, and for environmental and alveolar air from 0 to 1000 ppb. The total coefficient of variation for the two methods of measurement during the study was 6.5% for water and 18.5% for gaseous samples. Accuracy was verified by analyzing samples in an external, certified laboratory. The identity of chromatographic peaks was verified by mass spectrometry.

We established a correlation between alveolar and plasma CHCl₃ concentrations by plotting 20 paired measurements obtained from male subjects exposed to CHCl₃ (alveolar concentrations between 0 and 940 ppb). The Pearson coefficient (r^2) was 0.83, and the equation of the linear regression was CHCl₃(alveolar) = 29.6 CHCl₃(plasma) + 133.3 (Fig.1).

Maximum oxygen consumption (VO₂max) and the maximum heart rate (MHR) were measured using an activity test on a tread mill (Quinton-645, Washington). Oxygen supply (VO₂) was determined by an automated, open-circuit system using oxygen and carbon gas analyzers (model S-3A and Anarad AR-400, AMETEK, Pittsburgh, Pennsylvania). Ventilatory volumes were determined using a digital turbine spirometer (Model S-430, Vacumetrics/VACUMED Ltd., Venture, California) coupled to a 5.3-l

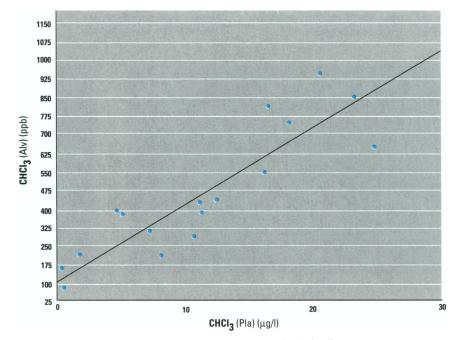


Figure 1. Simple linear regression between alveolar chloroform [CHCl₃(Alv)] and plasma chloroform [CHCl₃(Pla)]: CHCl₃ (alv) = 133.3 + 29.6 CHCl₃ (Pla), r^2 = 0.83.

mixing chamber. A qualified technician verified daily the calibration of the gas analyzers and the calibration of the turbine before each test. Heart rate was monitored by an ECG (Quinton-400, Seattle, Washington). Vo₂max was defined as the highest Vo₂ registered during the 1-min test.

To quantify the intensity of physical activity during the swimming exercise periods, the activity heart rate (AHR) for each swimmer was determined as a percentage, according to the MHR measured during the progressive tread mill test using the Dicarlo equation: AHR = $\% \times (MHR - 12)$ (22). During each exercise period, the AHR was determined every 15 sec using a heart rate monitor (PE 3000, Polar Electro, Finland) and verified every 5 min by a technician who gave advice to the subjects about their level of activity.

Using multiple regression models and a stepwise elimination process (19), we examined the influence of different variables on body burden at 35 min and 55 min: air and aqueous CHCl3 concentrations, the intensity of physical activity, and the physiological characteristics of the individuals. Data from day 6 (scuba equipment) were excluded from this analysis. Because the study design involved exposures of the same individuals to various concentrations, the subject was considered as a discrete variable in the model. This was necessary because of the interdependence of basic physiological characteristics (height, weight, etc.) in the same individual. The critical preselected value of the F distribution was established according to a 0.05 significance level. Since ambient air CHCl₃ were correlated to water concentrations, a separate regression model was used for each variable.

Results

Table 1 summarizes data on environmental CHCl₃ levels and the corresponding body burden in subjects. Prior to the seven exercise periods, before entering the swimming pool area, mean CHCl₃ concentration in alveolar air was 52.6 ppb (range 0-200 ppb, SD = 50.8 ppb) because of a slight air contamination in the locker room. Increasing the CHCl₃ concentrations in water and air from the swimming pool caused alveolar air concentrations to increase both at 35 and 55 min. Surprisingly, increasing basal (day 1) water concentration 1.4-fold on day 2 led to a similar increase (1.8-fold) in body burden at both 35 and 55 min. However, increasing aqueous CHCl₃ concentration from 159 μg/l on day 1 to 538 μg/l on day 7 (a 3.4-fold increase) led to a disproportionate 7.5-fold increase in body burden at 35 min and a 8.4-fold increase at 55 min.

Comparing data for day 2 to data for day 3 shows that despite the lower water concentration on day 3, an increase in the target intensity of exercise from 45% to 65% augmented the body burden of

CHCl₃ by 35% at 35 min and by 24% at 55 min. Unfortunately, the lack of air concentration data on day 2 (technical problems) is a serious drawback for properly assessing the influence of exercise intensity on body burden.

The proportion of CHCl₃ body burden that is due to inhalation can be obtained by comparing body burden data on day 5 with that collected on day 6, when the subjects used scuba tanks. During these two sessions, swimmers exercised at 45% of their maximum capacity, and aqueous concentrations were almost similar (552.8 ppb versus 567.5 ppb). For nine swimmers who participated in both sessions, average values for $\Delta 35$ and $\Delta 55$ were, respectively, 834.0 ppb and 996.0 ppb for day 5 and 195.6 ppb and 208.9 ppb for day 6 (dermal exposure route only). Hence, the average proportion of body burden that was due to inhalation route was 76% (SE = 3%) at 35 min and 78% (SE = 7%) at 55 min.

Given the strong influence of environmental CHCl₃ levels (water and air) on body burden, the relationship between In

Table 1. Chloroform (CHCl₃) body burden at 35 min (\triangle 35) and 55 min (\triangle 55) during the swimming pool exercise periods

Day	No. of swimmers	CHCl ₃ in water (µg/l)		CHCl ₃ in air (ppb)		∆35 (ppb)		∆55 (ppb)	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
1	9	158.6	7.5	507.0	45.8	100.0	23.8	103.9	23.8
2	9	229.3	6.7	8		179.4	105.9	185.6	57.1
3 ^b	11	200.0	10.7	1490.0	438.7	242.3	87.9	230.9	57.2
4	10	307.1	16.0	1120.0	157.1	283.0	116.4	276.5	101.6
5	11	552.8	34.5	1469.0	_c	950.0	303.2	1093.6	221.8
6 ^d	9	567.5	5.0	1296.0	36.5	195.6	53.6	208.9	185.5
7	10	538.3	21.4	1630.0	196.4	745.0	289.1	874.0	234.1

^aNo data available on air levels (day 2).

dExercise period with scuba tanks.

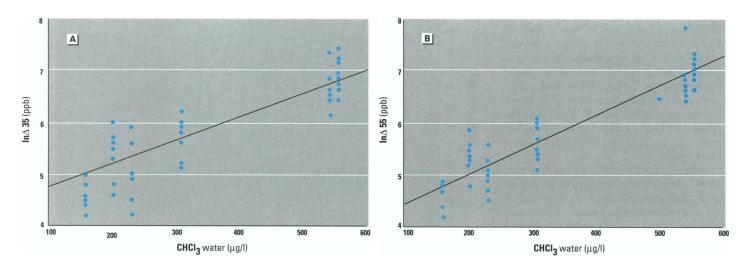
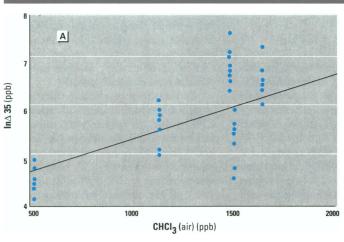


Figure 2. Simple linear regression between natural logarithm of the chloroform body burden at (A) 35 min and (B) 55 min and the chloroform concentration in water [CHCl₃ (water)]: $\ln \Delta 35 = 4.099 + 0.005$ CHCl₃ (water), $r^2 = 0.77$; $\ln \Delta 55 = 4.055 + 0.005$ CHCl₃ (water), $r^2 = 0.87$. Results from day 6 with scuba equipment were excluded.

^bIntensity of the physical activity at 65% maximum capacity.

^cOnly one result available (day 5).



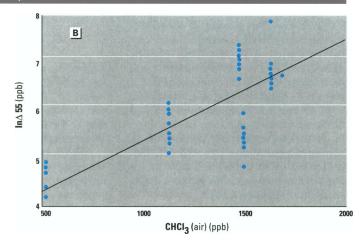


Figure 3. Simple linear regression between the natural logarithm of chloroform body burden at (A) 35 min and (B) 55 min and the concentration of chloroform in air [CHCl₃ (air)]: $\ln \Delta 35 = 3.591 + 0.002$ CHCl₃ (air), $r^2 = 0.59$; $\ln \Delta 55 = 3.58 + 0.002$ CHCl₃ (air), $r^2 = 0.60$. Results from day 6 with scuba equipment were excluded.

 $\Delta 35$ and $\ln \Delta 55$ and water concentrations (Fig. 2) or air concentrations (Fig. 3) were examined using simple linear regression analysis. Statistically significant relationships (p < 0.001) were observed between body burden indices and both water and air concentrations.

Multiple linear regression models that best explained variations of body burden at 35 and 55 min according to environmental CHCl₃ concentrations are described in Tables 2 and 3. The natural logarithm of the dependent variable $(\ln \Delta 35, \ln \Delta 55)$ was used to correct heteroscedasticity problems. This also allowed for a better description of the relationship with aqueous (Table 2) as well as environmental air (Table 3) CHCl₃ concentrations. For sake of clarity, B coefficients for the variable "subject," of which there are 11, i.e., one per "subject," were not included in Table 2 and 3. This variable was not statistically significant (p = 0.07) in the model describing the relationship between $\ln \Delta 35$ and environmental air CHCl₃ concentration (see Table 3).

Discussion

Table 1 indicates that Δ35 is essentially equivalent to Δ55 when aqueous CHCl₃ levels range between 158.6 ppb and 307.1 ppb, indicating that the equilibrium between elimination and absorption has been reached. However, at levels greater than 500 ppb (days 5 and 7), body burden seems to increase with duration of swimming. This might reflect saturation of biotransformation pathways and accordingly a longer half-life of elimination.

Table 2 presents relationships between aqueous CHCl₃ concentration and body burden using multiple linear regression models, for 35- and 55-min exposures. These models explained 91% and 95% of lnΔ35 and lnΔ55 variation, respectively, with water concentration alone accounting for 75% and 86%. Intensity of the physical activity as well as the "subject" are two other variables that explain a significant part of the body burden variation. Coefficients for aqueous CHCl₃ concentration and intensity of physical activity were similar in both lnΔ35 and lnΔ55 models.

Table 2. Stepwise multiple linear regression between the natural logarithm of the chloroform body burden at 35 min (Δ 35) and 55 min (Δ 55), and the concentration of chloroform in water

		In∆35		In∆55			
Independent variables	ß	р	r ²	ß	р	r ²	
CHCl ₃ in water ^a	0.005	<0.001	0.75	0.005	<0.001	0.86	
Intensity	0.024	< 0.001	0.04	0.020	< 0.001	0.03	
Subject	_	<0.001	0.12		<0.001	0.06	

^aResults from day 6 with scuba equipment were excluded.

Table 3. Stepwise multiple linear regression between the natural logarithm of the chloroform body burden at 35 min (Δ 35) and 55 min (Δ 55) and the concentration of chloroform in air

		In∆35		In∆55		
Independent variables	ß	р	r ²	ß	р	r²
CHCl ₂ in air ^a	0.002	<0.001	0.58	0.002	<0.001	0.63
CHCl ₃ in air ^a Intensity	-0.038	< 0.001	0.15	-0.053	< 0.001	0.14
Subject		_	_	_	0.04	0.07

^aResults from day 6 with scuba equipment were excluded.

Highly significant relationships were also noted between $\ln \Delta 35$ and $\ln \Delta 55$ and environmental air CHCl₃ levels (Table 3). Again, β coefficients were identical in both models. The variable "intensity" was also statistically significant, explaining up to 15% of body burden variation. The β coefficients were negative, suggesting that the intensity of the physical activity has a negative influence on the body burden. This contradicts models including aqueous CHCl₃ as an independent variable (see Table 2) and data from Table 1, both showing a positive correlation between intensity of exercise and body burden.

However, the models using aqueous CHCl₂ (Table 2) are likely more valid than those using ambient air CHCl₃ (Table 3). First, the variation of the dependent variable is better explained. Simple linear regressions illustrated in Figures 2 and 3 show that body burden at 35 and 55 min are better correlated with the aqueous CHCl₂ levels than with air concentrations. Second, precision of laboratory measurements of aqueous CHCl₃ is much greater than that of air samples (see Methods). In addition, the small-volume air samples (20 ml) collected periodically from the respiratory zone of the swimmers may have been contaminated by water droplets from splashing caused by the swimmers' movements (14). Continuous air sampling at 1.5 m from the water surface might have been preferable.

Aggazzotti and colleagues (14) recently evaluated alveolar CHCl₃ levels in 163 subjects (swimmers and nonswimming visitors) who spent 90 min in an indoor swimming pool. Environmental air CHCl₃ levels collected during eight sampling sessions in two indoor swimming pools explained 58.3% of the variation in alveolar CHCl₃ of subjects after exposure (14), a result similar to that obtained in the present study using the natural logarithm of body burden as a dependent variable (see

Table 3). However, contrary to our results, the aqueous CHCl₃ concentrations were not correlated to alveolar CHCl₃ levels. This is probably due to the limited range of aqueous CHCl₃ concentrations documented by Aggazzotti and co-workers (14), with all but one value between 159 and 369 nmol/l (19–44 μg/l).

Jo and co-workers (17,20) showed an average increase in alveolar air CHCl₃ concentration of 13 µg/m³ (2.7 ppb) and a correlation between alveolar air CHCl3 levels and aqueous CHCl₃ levels ($r^2 =$ 0.85) among 6 subjects who took 13 showers (10-min shower, 40°C water, use of soap and shampoo) (17,20). Using these results obtained under different exposure conditions, they showed a relationship similar to the one we demonstrated between aqueous CHCl3 and $\Delta 35$ and $\Delta 55$. However, Jo and colleagues attributed 48% of the total body burden to the dermal exposure route. Our results indicate that dermal absorption contributes much less (~25%) to the total body burden during swimming. This difference is possibly due to different exposure conditions. Taking a hot shower (40°C) produces an increased peripheral perfusion favoring absorption of CHCl3 by the skin. Likewise, use of soap and shampoo may be a contributing factor to the increased importance of the dermal exposure route.

Jo and colleagues (20) also compared the dose resulting from a 10-min shower to that obtained from ingesting 0.15–2.0 l of drinking water. To calculate the doses, they used previously documented water and air concentrations of 24 μg/l and 157 μg/m³ (32 ppb) (17) and the following set of equations:

$$D_i = E_r \times C_2 \times R \times T$$
/weight, (1)

where D_i = CHCl₃ dose from inhalation (µg/kg body weight); E_r = absorption efficiency of CHCl₃ by inhalation (0.77); C_a = shower air concentration (157 µg/m³; 32 ppb); R = breathing rate (0.014 m³/min); T = shower duration (10 min); and weight = 70 kg.

$$D_{\rm d} = D_{\rm i} \times F, \qquad (2)$$

where $D_d = \text{CHCl}_3$ dose from a dermal exposure (µg/kg boby weight); and $F = \text{ratio of the body burden from dermal exposure to body burden from inhalation exposure (0.93) (16).$

$$D_{i\sigma} = E_i \times C_w \times A_w / \text{weight}$$
, (3)

where D_{ig} = dose from water ingestion (µg/kg body weight); E_{i} = absorption efficiency of CHCl₃ via the gastrointestinal tract (100%); C_{w} = tap water concentration of CHCl₃ = 24 µg/l; and A_{w} = water amount ingested per day = (0.15–2.0 l).

The dose resulting from taking a 10-min shower was 0.46 μg/kg/day and that from ingestion of 0.15–2.0 l of drinking water varied between 0.05 μg/kg/day and 0.7 μg/kg/day (20).

Using the same rationale, we calculated doses associated with swimming in an indoor swimming pool, using CHCl₃ concentrations documented on the first day of our study, which are representative of levels frequently documented in public swimming pools (8-13). Assuming a breathing rate of 0.03 m³/min, equivalent to moderate physical activity (45% maximum capacity), and a ratio of body burden from dermal exposure to that from inhalation exposure of 0.30, we estimated at 65 µg/kg/day the total CHCl3 dose from a 1hr swim (50 µg/kg/day for inhalation exposure and 15 µg/kg/day for dermal exposure). Under these conditions, CHCl₃ dose resulting from a 1-hr swim (65 µg/kg/day) is 141 times greater than that for a 10-min shower (0.46 µg/kg/day) and 93 times greater than exposure by tap water ingestion as demonstrated by Jo and co-workers (17). This conclusion is supported by the large difference between body burden from showering (2.7 ppb) (17) and that resulting from 55 min of swimming (103.9 ppb) during day 1 of our study.

Our results suggest that THM standards for drinking water (5,21,22) (Table 4), which are based solely on ingestion exposure, without taking into consideration other routes of exposure, may not afford protection to all segments of the population, especially swimmers. However, the concept of dose as described above is probably too simplistic for correctly evaluating the risk associated with each route of exposure. Target organ concentrations are more suitable for risk assessment and will differ according to the exposure route. A pharmacokinetic model, such as that described by Corley et al. (23), may be useful for estimating target organ concentrations resulting from multimedia exposure. With more information about various physiological variables (height, weight, fat %), data from our study, which links water and air concentrations to body burden (alveolar CHCl₃), may certainly be used to further refine and validate such a model.

Our study shows clearly that swimming is an important source of exposure to CHCl₃, through both inhalation and dermal routes. Using the relationship between InΔ55 and CHCl₃ concentration in swimming pool water (see Fig. 2), body burdens corresponding to 100 µg/l, 200 µg/l, and 400 µg/l are, respectively, 95 ppb, 157 ppb, and 426 ppb. Although this relationship will be influenced by pool size and ventilation rate, results obtained are representative of the prevailing conditions in many other similar indoor swimming pools. It may be possible to exert tighter controls on chlorination practices, thereby decreasing the formation of chlorination by-products, including CHCl₃, while still preserving microbiological water quality. Ozonation and electronic purification devices may also prove to be valid alternatives.

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Agency	Standard (µg/l)	Reference
World Health Organization (recommendation for chloroform only)	200	(5)
U.S. Environmental Protection Agency (maximum annual average value)	100	(21)
Health and Welfare Canada (maximum value)	350	(22)

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International Symposium on Environmental Biomonitoring and Specimen Banking

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